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L10 ANSWER 1 OF 41 MEDLINE
AN 2000264937 MEDLINE
DN 20264937
TI Invasive examination of cardiovascular disease.
AU Horimoto M; Takenaka T; Igarashi K; Inoue H; Akino M
CS Division of Cardiology, Sapporo National Hospital.
SO RINSHO BYORI. JAPANESE JOURNAL OF CLINICAL PATHOLOGY, (2000 Feb) 48 (2) 128-37. Ref: 28
Journal code: KIV. ISSN: 0047-1860.
CY Japan
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW LITERATURE)
LA Japanese
EM 200009
EW 20000902

16504/94

L10 ANSWER 5 OF 41 MEDLINE
AN 1999242350 MEDLINE
DN 99242350
TI Short-term and long-term effects of low-density lipoprotein (LDL) apheresis on restenosis after percutaneous transluminal coronary angioplasty (PTCA): is lowering Lp(a) by LDL apheresis effective on restenosis after PTCA?.
AU Kanemitsu S; Takekoshi N; Matsui S; Tsugawa H; Ohkubo S; Kitayama M; Matsuda T; Senma J; Masuyama K; Yamagata T; Murakami E
CS Department of Cardiology, Kanazawa Medical University, Kahoku-gun, Ishikawa-ken, Japan.
SO Ther Apher, (1998 Feb) 2 (1) 65-70.
Journal code: DBB. ISSN: 1091-6660.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199907
EW 19990703

L10 ANSWER 6 OF 41 MEDLINE
AN 1999240278 MEDLINE
DN 99240278
TI Clinical application and effectiveness of low-density lipoprotein apheresis in the treatment of coronary artery disease.
AU Daida H; Yamaguchi H
CS Department of Cardiology, Juntendo University School of Medicine, Tokyo, Japan.
SO Ther Apher, (1997 Aug) 1 (3) 253-4.
Journal code: DBB. ISSN: 1091-6660.
CY United States
DT (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals

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Therapeutic Apheresis
1(3):253–254, Blackwell Science, Inc.
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Clinical Application and Effectiveness of Low-Density Lipoprotein Apheresis in the Treatment of Coronary Artery Disease

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Abstract: The Low-Density Lipoprotein Apheresis Coronary Atherosclerosis Prospective Study (L-CAPS) examined whether or not combined low-density lipoprotein (LDL) apheresis and drug therapy apheresis could induce the regression of coronary atherosclerotic lesions in patients with familial hypercholesterolemia. Twenty-eight patients treated with LDL apheresis and drugs and 11 patients treated with drugs alone underwent sequential coronary angiography 2.5 years apart. The frequency of cases with regression or no change was significantly higher for the apheresis group than for the control group ($p = 0.004$). The LDL apheresis Angioplasty Restenosis Trial (LART) investigated the hypothesis that high plasma lipoprotein (a) (Lp[a]) levels were associated with increased incidences of restenosis after coronary angioplasty. Two days before and 5 days after angioplasty, 66 patients underwent LDL apheresis. The restenosis rates were 21% in the 42 patients whose Lp(a) levels were reduced ≥ 50% and 50% in the 24 patients whose Lp(a) levels were reduced < 50% ($p < 0.05$). LDL apheresis is effective in the prevention of the progression of coronary atherosclerosis. Its potential application in restenosis prevention should be further investigated. **Key Words:** Low-density lipoprotein apheresis—Progression of coronary atherosclerosis—Lipoprotein (a)—Restenosis.

The low-density lipoprotein (LDL) apheresis using the Liposorber system (Kaneka Corporation, Tokyo, Japan) is a unique technology that selectively removes apoprotein B containing lipoprotein (1). It has been widely used in Japan for the long-term treatment of familial hypercholesterolemia (FH) (2,3). There have been studies that investigated other applications of this new technology. We present the results of 2 recent trials, the Low-Density Lipoprotein Apheresis Coronary Atherosclerosis Prospective Study (L-CAPS) (4) and the LDL-Apheresis Angioplasty Restenosis Trial (LART) (5).

The L-CAPS examined prospectively whether combined LDL apheresis and lipid lowering drugs could induce the regression of coronary atherosclerotic lesions in patients with familial hypercholesterolemia (FH). Twenty-eight patients treated with LDL apheresis and drugs (apheresis group) and 11 patients treated with drugs alone (control group) underwent sequential coronary angiographies 2.5 years apart. Coronary angiograms were analyzed quantitatively in 85 segments of the apheresis group and in 51 segments of the control group. The total cholesterol levels in the plasma were reduced from 317 ± 92 mg/dl to 171 ± 30 mg/dl in the apheresis group and from 339 ± 15 mg/dl to 242 ± 67 mg/dl in the control group. The mean LDL-cholesterol levels during the trial were 122 ± 32 mg/dl in the apheresis group and 176 ± 64 mg/dl in the control group ($p < 0.05$). The minimal lumen diameter in the apheresis group increased from 1.85 ± 0.74 mm to 2.02 ± 0.77 mm while that in the control group decreased from 2.30 ± 1.00 to 1.92 ± 0.77 mm ($p < 0.0005$). In the apheresis group, 5 cases showed regression (18%), 2 showed progression (7%), and 20 stayed unchanged (71%). In contrast, 6 cases showed progression, 5 cases stayed unchanged (45%), and there were no regression cases in the control group. The frequency of cases with regression or no change was significantly higher in the apheresis group than in the control group ($p = 0.004$).

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LDL apheresis has been shown to reduce plasma lipoprotein (a) (Lp [a]) (6), which is a possible risk factor for restenosis after percutaneous transluminal coronary angioplasty (7). The LART investigated the hypothesis that high plasma Lp(a) levels were associated with the increased incidence of restenosis after coronary angioplasty. Two days before and 5 days after angioplasty, 66 patients underwent LDL apheresis; 39 patients also received 10 mg of pravastatin and 1,500 mg of niacin daily. The median Lp(a) levels were reduced from 23.3 mg/dl to 10.9 mg/dl ($p < 0.0001$). Restenosis occurred in 38% of the 137 control patients and in 32% of the 66 patients who underwent LDL apheresis (NS). The restenosis rates were 21% in the 42 patients whose Lp(a) levels were reduced $\geq$ 50% and 50% in the 24 patients whose Lp(a) levels were reduced < 50% ($p < 0.05$). Among the patients who received pravastatin and niacin in combination with apheresis, the restenosis rate was 12.5% in the 24 patients whose Lp(a) levels were acutely lowered, that is, $\geq$ 50% and 53% in the 15 patients for whom the acute reductions of Lp(a) levels were < 50%.

In conclusion, intensive cholesterol lowering by LDL apheresis was effective in preventing coronary atherosclerosis progression, and it could induce the regression of atherosclerosis in patients with FH. Its potential application in restenosis prevention by means of Lp(a) reduction after coronary angioplasty should be further investigated.

Acknowledgment: The authors have submitted this paper on behalf of the Low-Density Lipoprotein Apheresis Coronary Prospective Study (L-CAPS) and Low-Density Lipoprotein Apheresis Angioplasty Restenosis Trial (LART) groups.

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